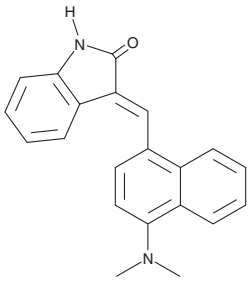
PRODUCT INFORMATION

MAZ51

Item No. 16168

CAS Registry No.:	163655-37-6
Formal Name:	3-[[4-(dimethylamino)-1-naphthalenyl] methylene]-1,3-dihydro-2H-indol-2-one
MF:	$C_{21}H_{18}N_2O$
FW:	314.4
Purity:	≥95%
UV/Vis.:	λ_{max}: 210, 260, 405 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

MAZ51 is supplied as a crystalline solid. A stock solution may be made by dissolving the XX in the solvent of choice, which should be purged with an inert gas. MAZ51 is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of MAZ51 in these solvents is approximately 1 and 0.2 mg/ml, respectively.

Description

The VEGF receptors (VEGFRs) mediate signaling leading to angiogenesis, with VEGFR3 (FLT4), activated by VEGF isoforms VEGFC and VEGFD, primarily expressed on lymphatic endothelial cells and directing lymphangiogenesis.[1] MAZ51 is an indolinone that selectively antagonizes the activation of VEGFR3 by VEGFC (IC_{50} = 1 µM) without blocking VEGFC-mediated stimulation of VEGFR2.[2] It does not inhibit ligand-induced autophosphorylation of EGFR, IGF-1R, or PDGFRβ.[3] By preventing VEGFR3 activation, MAZ51 interferes with an autocrine loop involving the induced expression of the ligand VEGFC as well as VEGFA.[4] It reduces proliferation and induces apoptosis in a variety of cancer cells *in vitro* and suppresses tumor growth *in vivo*.[3] MAZ51 is used to study the role of VEGFR3 in endothelial and cancer cell function and survival.[5,6]

References

1. Manley, P.W., Bold, G., Brüggen, J., *et al.* Advances in the structural biology, design and clinical development of VEGF-R kinase inhibitors for the treatment of angiogenesis. *Biochim. Biophys. Acta* **1697(1-2)**, 17-27 (2004).
2. Kirkin, V., Mazitschek, R., Krishnan, J., *et al.* Characterization of indolinones which preferentially inhibit VEGF-C- and VEGF-D-induced activation of VEGFR-3 rather than VEGFR-2. *Eur. J. Biochem.* **268(21)**, 5530-5540 (2001).
3. Kirkin, V., Thiele, W., Baumann, P., *et al.* MAZ51, an indolinone that inhibits endothelial cell and tumor cell growth *in vitro*, suppresses tumor growth *in vivo*. *Int. J. Cancer* **112(6)**, 986-993 (2004).
4. Matsuura, M., Onimaru, M., Yonemitsu, Y., *et al.* Autocrine loop between vascular endothelial growth factor (VEGF)-C and VEGF receptor-3 positively regulates tumor-associated lymphangiogenesis in oral squamoid cancer cells. *Am. J. Pathol.* **175(4)**, 1709-1721 (2009).
5. Park, J.H., Shin, Y.J., Riew, T.R., *et al.* The Indolinone MAZ51 Induces Cell Rounding and G2/M Cell Cycle Arrest in Glioma Cells without the Inhibition of VEGFR-3 Phosphorylation: Involvement of the RhoA and Akt/GSK3ß Signaling Pathways. *PLoS One* **9(9)**, 1-9 (2014).
6. Breslin, J.W., Yuan, S.Y., and Wu, M.H. VEGF-C alters barrier function of cultured lymphatic endothelial cells through a VEGFR-3-dependent mechanism. *Lymphat. Res. Biol.* **5(2)**, 105-113 (2007).

CAYMAN CHEMICAL
1180 EAST ELLSWORTH RD
ANN ARBOR, MI 48108 · USA
PHONE: [800] 364-9897
[734] 971-3335
FAX: [734] 971-3640
CUSTSERV@CAYMANCHEM.COM
WWW.**CAYMANCHEM**.COM